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Studies in Experimental Scurvy of the Guinea Pig

STUDIES IN EXPERIMENTAL SCURVY
OF THE GUINEA PIG

BY

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I HEREBY RECOMMEND THAT THE THESIS PREPARED UNDER MY

SUPERVISION BY DORIS VON EISEN BOCKIUS

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TABLE OF CONTENTS.

I. GENERAL INTRODUCTION.

Scurvy, a disease occasioned by the absence of green foods from the diet and characterized particularly by a hemorrhagic condition of the soft parts of the bones and gums, has been recognized for a long time. The study of both the human and experimental types has been developed along three lines; first, the dietary factors which influence the progress of the disease, second, the anatomical changes produced by the disease, and third, the chemical changes in the body tissues and fluids during scurvy. Most of the work to date has been along the first two lines. In view of the marked pathological changes which accompany scurvy it seems highly probable that decided alterations in the composition of the body and its fluids must also occur. Yet, only recently have any investigations been made on the chemical phases of the disease. The object of this paper was to obtain any additional information possible concerning the chemical changes which might occur in the body during scurvy.

II. HISTORICAL.

To Funk[1], in 1912, we owe the term vitamine which he employed to denote the unknown essential principles whose absence from the diet causes beriberi. The term has, however, become established in general use to signify any accessory substance necessary for satisfactory metabolism, the lack of which in a diet will lead to the occurrence of a "deficiency" disease. Scurvy is generally considered to be one of these deficiency diseases. The work on deficiency diseases was begun in 1897 by Eijkmann[2] when he observed that beriberi was due to a one-sided diet of polished rice. Further study by many investigators has established without question that beriberi is due to a deficiency in the diet. Casimir Funk[1] first used the term"deficiency diseases"as applied to the following, beriberi, polyneuritis in birds, epidemic dropsy, scurvy, experimental scurvy, infantile scurvy, ship-beriberi and pellagra. He believed in the existence of a number of essential chemical substances in the diet, the absence of any one of which would cause abnormal metabolism, polyneuritis, scurvy, pellagra or rickets according to the type of deficiency.

It has long been recognized that scurvy is a nutritional disease, and although the cause of it is still rather obscure, the means of cure has been known for sometime. The first observers believed that the disease was due to infection, to toxemia, or to acidosis[3].

The first work on experimental scurvy was that of Theobald Smith[4], who in 1895 reported that guinea pigs died of a peculiar hem-

[illegible]

[illegible]

[illegible]

[illegible]

orrhagic disease when fed on a cereal diet without succulent vegetables. To Holst and Frölichs, in 1907, however, we are indebted for the first recognition of the identity of the disease resulting from an exclusive cereal diet in guinea pigs with human scurvy or ship-beriberi. Later Holst and Frölich continued their work and found that an unvarying diet of grain or bread caused, in guinea pigs, a disease which in all pathological and anatomical relations resembled scurvy in man. They were able to produce this disease with almost mathematical certainty, when guinea pigs were fed on an exclusive diet of oats or other grains. In the course of 28 to 30 days the animals failed and died. Autopsy showed a loosening of the back teeth, hyperemia of the gums, hemorrhages in the muscles about the joints of the extremities, and hemorrhages about the costochondral junctions, and frequently epiphysolitis about the upper end of the tibia. Further, the histological changes in the epiphyses and in the bone marrow were identical with those seen in infantile scurvy They failed to produce scurvy in guinea pigs by feeding with heated milk, unless it were heated to a higher temperature than that used in infant feeding. They were unable to explain this difference between experimental scurvy and infantile scurvy. Further work showed that milk heated above 100° C. did not produce the disease, but lost to some degree its power to cure it. They found, also, that a constant diet of certain fresh vegetables, cabbage, carrots, dandelions, and others, resulted in a loss of weight but caused no scorbutic symptoms similar to those observed on a grain diet. These vegetables possessed antiscorbutic properties, i.e., their administra-

tion to the affected guinea pigs resulted in recovery from the scurvy.

Drying and heating, as a rule, reduced or destroyed the antiscorbutic properties. The formation of antiscorbutic substances could be demonstrated in the process of germination of grains, as was later confirmed by McClendon[6] and others. Holst and Frölich believed that the disease was not caused by acidosis, but was due to an insufficient supply of certain substances of unknown chemical nature, present in the normal diet. The nature of the action of these antiscorbutic substances was unknown.

Fürst[7] did work along the same lines and found that a diet of dry plant seeds resulted in cases of scurvy milder than those produced by the diets of grains. He believed that, while in the end death resulted from under-nurishment, this was not the cause of the disease, and drew conclusions as to its etiology, which were similar to those of Holst and Frölich[5]. Hart[8] was able to produce the characteristic bone changes in guinea pigs, fed on milk alone. With Talbot[9] he also succeeded in producing scurvy in monkeys.

The next important investigators are McCollum and Pitz[10]. From their work on scurvy they concluded that it was not a deficiency disease proper, but resulted from the impaction of the colon occasioned by the constipating character of the scorbutic diet. It was suggested that the scorbutic syndrome was due either to absorption of toxic products of bacterial origin from the intestine, or to mechanical injury of the intestional wall, which permitted bacterial invasion of the system. They considered that the beneficial action of orange juice was due to its laxative properties. Gerstenberger[11]

however, has recently shown that orange juice may be constipating in its action. McCollum and Pitz also contended that polyneuritis, the disease in birds due to lack of water-soluble B was the only deficiency disease.

On the contrary, the more recent work seems to give full support to the earlier views of Holst and Frölich$_{8}$, that scurvy is a result of a deficiency of some nutritive factor in the diet. As pointed out by Chick and Hume$_{12}$, McCollum's$_{10}$ conclusions were based on data obtained from experiments where a varying factor was constantly present, in that all the experimental animals were allowed milk ad libitum. As a result, the amount of milk taken by each guinea pig depended to a great extent, not only on the individual idiosyncracies of each animal but also on the changes in experimental conditions which tended to modify the liquid intake. It has further been shown by Chick and Hume, that the injection of 50 to 100 cc. of milk will provide some protection against scurvy, whereas 100 to 150 cc. will entirely prevent scurvy.

Further work has been done in favor of the deficiency theory by Hess$_{13}$ who found that injection of orange juice would prevent scurvy. This seemed to prove the presence of a vitamine and to minimize the intestional factor on which McCollum and Pitz$_{10}$ placed so much emphasis. Hess and Unger$_{14}$ found that guinea pigs fed on oats, hay, and water, were not constipated, nor was the cecum of those dying of scurvy found to be impacted, but contained a gas and semisolid feces. Torrey and Hess$_{15}$ also noted no difference in the intestional flora of normal and scorbutic guinea pigs. Cohen and Mendel$_{16}$ showed that scurvy was not essentially dependent upon constipation as a causative factor, tho they believed the latter may aggravate the symptoms.

III. CHEMICAL CHANGES IN DEFICIENCY DISEASES.

Very recently considerable work has been done on the chemical changes that take place in the body in the deficiency diseases.

Excessive salivation is commonly associated with pellagra. Sullivan[17] who has studied the saliva in this disease believed that this was often more apparent than real and was seemingly due to some inhibition of swallowing combined with a peculiar ropy change in the saliva, a high content of mucus, which made the presence of saliva in the mouth more obvious. In unpublished experiments he reports no changes in the alkaline reserve in pellagra. Still other chemical changes in pellagra have been investigated by Koch and Voegtlin[18]. They found that, in a general way the spinal cord exhibits the most striking chemical changes, a fact which is in agreement with histological observations. These changes seem to involve principally certain lipoids and also cause an increase in cholestrol in the cerebellum and spinal cord, and a decrease in that substance in the cerebrum.

In scurvy the most marked among these chemical changes has been the alterations in the adrenals, and adrenaline content of the adrenals, in such diseases as beriberi and scurvy. Robert McCarrison[19] has observed the following changes in beriberi;: first, an enlargement of the adrenals and secondly, an adrenaline content of the glands increased above normal. Like analyses of the adrenals of patients dying from beriberi have shown similar results. On the contrary no changes were found in the adrenals during rickets. Robert McCarrison[20] has done similar experiments on scurvy in which he found an

increase in the size and weight of the adrenal glands and a marked decrease in the adrenaline content of these glands, as compared with the control guinea pigs. In his experiments the basal diet for the controls was not comparable to that of the scorbutic pigs, thus making his results difficult of interpretation. Hemorrhagic infiltrations and degenerative changes in the cellular elements of the adrenal cortex and medulla were also noted.

Other chemical changes have been demonstrated to occur in the scorbutic guinea pig. Lewis and Karr[21] found that the urea content of the blood of scorbutic guinea pigs was several times higher than the normal amount. Hess and Killian[22], however, observed no alteration in the urea content of the blood in infantile scurvy. Nor did they find a change in the blood sugar, but found a moderate acidosis and a deficiency of calcium. Neither of these last two appeared to be a basic factor, or to run a course parallel to the scorbutic process. Zilva and Wells[23] observed that scurvy produced a fibrosis or fibroid degeneration of the dental pulp. The tooth was one of the first, if not the first part of the system to be affected by deficiency of antiscorbutics in the diet. They noted profound changes in the teeth when the scorbutic symptoms during life had been so slight as to be almost unrecognizable. A diet deficient in antiscorbutics produced radical changes in the teeth of the monkey also. McClendon, Cole, Engstrand, and Middlekauff[6] were able to detect no changes in the alkaline reserve of the blood of scorbutic guinea pigs and concluded acidosis had no relation to the pathology of the disease, a conclusion in harmony with the work of Funk[24]. Baumann and Howard[25] have done considerable work on the mineral metabolism

in experimental scurvy of the guinea pig, and believe that the lack of antiscorbutic substances in the diet have a profound effect on the mineral metabolism of the guinea pig. The most marked changes, they found, were increased losses of calcium and phosphorous. Lewis and Karr̸e obtained no changes in the urinary elimination of phenols in guinea pigs on an oat diet, nor in the degree of conjugation of the phenols, provided the factor of partial starvation was ruled out. This they believed to indicate that no increased bacterial action occurs in the intestine of the scorbutic guinea pigs despite the difficulty of evacuation of the feces. In the present paper a study has been made of the adrenaline content of the adrenals, the glycogen content of the liver and the blood sugar in scorbutic guinea pigs and normal controls.

IV. EXPERIMENTAL.

For the experimental work young guinea pigs, of 200 to 300 grams weight, were used. They were kept under observation on a normal mixed diet for a period of two weeks before beginning the experiment. Special precautions were taken to prevent infection. The cages were scrubbed daily with frequent use of carbolic acid and other disinfectants. The guinea pigs were kept in separate cages and their body weight and food consumption were recorded daily. All of the pigs received water ad libitum and a basal diet of from 10 to 30 grams of an oat cake of the following composition:

Autoclaved rolled oats	500 grams
Wheat bran	100 grams
Purified casein	40 grams
Calcium lactate	15 grams
Sodium Chloride	15 grams
Whole milk	200 cc.
Distilled water	400 cc. ±

The rolled oats were heated in an autoclave for 30 minutes at 120°. The dry ingredients were thoroughly mixed and the milk and water added. The pasty mass was spread in thin layers on pie tins and dried at 75 - 80°. This formed a cake of hard enough consistency to prevent its being scattered around the cage. The controls received the same basal diet with the addition of 10 to 15 grams of fresh orange daily.

For the determination of adrenaline in the adrenals the method of Folin, Cannon, and Denis[27] was used. The method is based upon the quantitative production of blue color when a solution of adrenaline

is treated with phosphotungstic acid and sodium carbonate. Uric acid and other substances likewise give the reaction, but can be disregarded when the determinations are made upon adrenal glands. The determination of the glycogen in the liver was made according to Pflüger's[23] method, the glucose being determined gravimetrically by Allihn's modification of Fehling's method. For the determination of blood sugar the Benedict modification of the method of Lewis and Benedict was used.

V. RESULTS.

1. Glycogen of the Liver.- Analysis of the liver showed a decrease in the content of glycogen in the scorbutic guinea pig as compared with the control. It is commonly known that the glycogen of the liver is markedly decreased in starvation. Altho as shown in the protocols of the appendix, the food intake was markedly decreased as the scorbutic condition progressed, yet in no case was there complete fasting. A condition of partical starvation was undoubtedly present and may be regarded as the cause of the decrease in the glycogen of the livers of the scorbutic pigs, rather than the disease itself. The relative importance of the two factors, partial starvation and scorbutus, cannot be estimated from the available data.

2. Blood Sugar.- The sugar content of the blood was slightly increased in the scorbutic animals as compared with the controls. However, the scorbutic guinea pig receives an insufficient supply of water, since the animals, which normally receive their water in the form of succulent vegetables, in many cases refuse to drink from the water vessels. It has been shown by Lewis and Karr[21] that the increase in the urea content of the blood of scorbutic animals may be diminished by the forced ingestion of water. They consider that de-

hydration of the system is one factor in promoting the accumulation of urea in the system. It is possible, in the case of the blood sugar, that the same factor may also be effective, altho it cannot be stated definitely that the disease is not also responsible in part for the changes.

3. Adrenaline.- Macroscopically the adrenals were found to be larger in the scorbutic guinea pigs than in the controls. The average weight of the glands in the scorbutic animal was 0.2235 grams as compared with that of the normal pig which was 0.1866 grams. This difference becomes more marked when the weight of the glands is calculated per kilo of original and of final body weight. The average weight of adrenal per kilo of original body weight in the scorbutic pig was 1.0078 grams as compared with 0.7567 grams for the normal pig, while if the calculations are made on the basis of final body weight the figures are 1.2041 grams and 0.5954 grams respectively. It is considered that it is more reasonable, however, to compare the total adrenal tissue per kilo of final body weight of the normal control with that per kilo of the original body weight of the scorbutic guinea pig, rather than with that of the final body weight of the latter. The control animals were rapidly increasing in body weight throughout the entire period and it is reasonable to assume an increase in the weight of the adrenals in proportion to the somatic development. On the other hand, altho the scorbutic pigs decreased in weight starvation is generally not held to affect the more essential organs, glands, etc., until it has progressed nearly to the point of death. The weight of the adrenals then, if starvation alone were involved, might have been expected to be proportional to the

original body weight. For this reason comparison of the weight of the adrenals in terms of the original body weight of the scorbutic animals with that of the same organs in terms of final body weight of the controls seemed fairer. Despite any possible atrophy of gland due to partial starvation, this comparison shows a greater weight of adrenal tissue in the scorbutic guinea pigs than in the controls. As for the adrenaline present, the amount in the scorbutic animals varied from 0.0196 mg. to 0.0386 mg. with an average of 0.0287 mg. as contrasted with that of the controls which varied from 0.0435 mg. to 0.1017 mg. with an average of 0.0692 mg., even tho the glands in the former were considerably larger than those in the controls. A comparison of the amount of adrenaline per kilo of original body weight in the scorbutic guinea pigs with that in the controls shows a decided decrease in the former. The average weight of adrenaline per kilo original body weight in the scorbutic animal was 0.12 mg., while that in the controls was 0.28 mg. This decrease is not so marked when the relation is taken to the final body weights rather than to the original body weights. The average weight of adrenaline per kilo final body weight in the scorbutic guinea pigs was 0.15 mg. in comparison with that of the controls which was 0.23 mg. This shows that the total adrenaline does not diminish in proportion to the decrease in the body weight. In a comparison of the weight of adrenaline, per gram of the adrenal, the controls were found to have more than three times as much as the scorbutic animals. These results are in accord with the findings of McCarrison[20], altho the differences observed are not as great as those observed by him. There was found to be no direct relation

between the alterations in the weight and adrenaline content of the adrenals and the duration of the disease. This can be shown by a comparison of guinea pigs (5) and (12). The duration of the experiment with the former was 15 days, the adrenals weighed 0.2619 grams, and the adrenaline content was 0.0299 grams, whereas the experimental period in the case of the latter was 21 days, the adrenals weighed only 0.2034 grams, with an adrenaline content of 0.0386 grams. No relation could be determined between the change in weight and adrenaline content of the adrenals and the food intake. This is well illustrated by pigs (10) and (12) in which the duration of the experiment was the same. In the case of pig (10) the adrenals weighed 0.2128 grams and contained 0.0301 grams adrenaline, while in the case of pig (12) the adrenals weighed 0.2034 grams and contained 0.0386 grams adrenaline. In comparing their food intakes, the average for the last 7 days was, in the former, 10.5 grams and in the latter 15.4 grams. Also, there was found to be no difference between the adrenaline content of the adrenals which were analyzed immediately after death and those in which several hours elapsed before analysis was made. A comparison of pigs (5), (6), and (7) brings out this point. Both (5) and (6) were killed and analyses made immediately, whereas (7) died and analysis was not made until a couple of hours latter. The weight and adrenaline content of the adrenals were as follows:-

Pig 5 adrenals weighed .2691 gms. and contained .0298 gms. adrenaline
Pig 6 adrenals weighed .2103 gms. and contained .0378 gms. adrenaline
Pig 7 adrenals weighed .2258 gms. and contained .0322 gms. adrenaline

The results for pig (8) were low due to a loss of the adrenal tissue in removing the adrenals from the body.

No.	WEIGHT		DAYS	WEIGHT OF LIVER	GLYCOGEN IN LIVER		BLOOD SUGAR
	INITIAL gm	FINAL gm		gm	gm	%	%
				SCURVY			
1	165	150	8	5.30	Traces	- -	- -
2	175	161	8	5.20	.084	1.62	- -
3	206	183	8	6.83	Traces	- -	- -
4	254	200	14	6.27	Traces	- -	- -
5	280	229	15	9.64	.075	.78	.126
6	234	197	16	- -	- -	- -	.125
7	238	163	17	6.32	Traces	- -	- -
10	238	182	21	5.75	Traces	- -	- -
11	269	199	21	7.25	.031	.43	- -
12	258	244	21	13.22	.134	1.01	.117
				CONTROLS			
13	280	344	17	16.19	.559	3.45	.096
14	255	322	23	10.38	.147	1.35	.084
15	269	322	24	10.95	.194	1.77	.102
17	207	232	30	12.07	.488	4.04	- -

SHOWING THE ADRENALIN CONTENT OF THE ADRENAL GLANDS, THE TOTAL ADRENALIN PER KILO BODY WEIGHT, ETC.

Number of Guinea Pigs	Original weight of Guinea Pigs in Grams	Highest weight of Guinea pig in Grams	Final weight of Guinea pig in Grams	Days	Weight of Adrenals in Grams	Total Adrenalin in both Adrenal Glands in mgms	Weight of Adrenals per kilo of original body weight in gms.	Weight of Adrenals per kilo of final body weight in gms.	Total Adrenalin per kilo of original body weight in mgms.	Total Adrenalin per kilo of Final body wt. in mgms.	Total Adrenalin per Gram of gland
					SCURVY						
1	164	173	150	8	.1700	.0244	1.0365	1.1333	.15	.16	.14
2	175	178	160	8	.1914	.0197	1.0937	1.1920	.11	.12	.10
3	206		183	8	.1667	.0208	.8092	.9109	.10	.11	.12
4	254	263	200	14	.2766	.0330	1.0889	1.3830	.13	.17	.12
5	280	296	229	15	.2691	.0298	.9610	1.1751	.11	.13	.11
6	234	249	197	16	.2103	.0378	.8987	1.0675	.16	.19	.10
7	238	269	163	17	.2258	.0322	.9487	1.3852	.14	.20	.14
8	213	242	180	18	.1672	.0196	.7849	.9289	.09	.11	.12
9	209	224	161	19	.2590	.0317	1.2392	1.6086	.15	.20	.12
10	238	243	182	21	.2128	.0301	1.1256	1.1692	.13	.17	.14
11	269	304	199	21	.3306	.0268	1.2290	1.6613	.10	.13	.08
12	258	317	244	21	.2034	.0386	.7883	.8336	.15	.16	.19
					CONTROL						
13	280		344	17	.221	.0673	.7892	.6424	.24	.20	.30
14	255	325	322	23	.2239	.1017	.8780	.6953	.40	.32	.45
15	269		322	24	.1556	.0617	.5784	.4832	.23	.19	.40
16	225	349	349	30	.1844	.0435	.8195	.5163	.19	.13	.24
17	207	238	232	30	.1485	.0720	.7174	.6400	.35	.31	.48

VI. SUMMARY.

1. In scorbutic guinea pigs an increase in size and weight of the adrenal glands was observed.

2. Despite the increased weight of the adrenal glands, the total adrenaline content was decreased both absolutely and in proportion to the weight of the glands.

3. The glycogen content of the liver was diminished. It was not possible to determine to what extent, if any, this may have been due to partial starvation.

4. The blood sugar content was increased. This may have been associated with increased concentration of the blood due to insufficient water intake.

5. Changes in the composition of the adrenals may occur before marked clinical evidences of scurvy manifest themselves.

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Pig 1. Died March 25. Autopsy showed slight hemorrhages at knee joints.

Pig 2. Died March 25. Autopsy showed slight hemorrhages at knee joints.

Pig 3. Died March 25. Autopsy showed slight hemorrhages at knee joints.

Pig 4. Died May 4. Autopsy showed hemorrhages at knee joints.

Pig 5. Was killed May 5. Autopsy showed hemorrhages at knee joints and at costochondral junctions.

Pig 6. Was killed May 6. Autopsy showed hemorrhages at knee joints, at costochondral junctions, and also subcutaneous hemorrhages.

Pig 7. Died April 3. Autopsy showed hemorrhages at knee joints and subcutaneous hemorrhages.

Pig 8. Died April 4. Autopsy showed subcutaneous hemorrhages and hemorrhages in the costochondral junctions and knee joints.

Pig 9. Died April 5. Autopsy showed hemorrhages in costochondral junctions and knee joints. The leg bones were very brittle.

Pigs 10, 11, and 12. Died May 11. Autopsy showed marked hemorrhages in costochondral junction and knee joints. Leg bones were very brittle.

Pigs 13, 14, 15, 16, and 17 were all killed. Autopsy showed them to be normal.

DATE	BODY-WEIGHT	OAT-CAKE	DATE	BODY-WEIGHT	OAT-CAKE	DATE	BODY-WEIGHT	OAT-CAKE	DATE	BODY-WEIGHT	OAT-CAKE
	gms	gms		gms	gms		gms	gms		gms	gms
	PIG 1		4/28	244	8	5/5	222	14	4/2	194	15
3/17	165	--	4/29	228	11	5/6	196	8	4/3	188	15
3/18	---	10	4/30	241	10		PIG 7		4/4	180	10
3/19	173	9	5/1	229	13	3/17	238	--		PIG9	
3/20	---	14	5/2	209	15	3/18	---	17	3/17	209	7
3/21	167	12	5/3	201	7	3/19	265	25	3/18	---	14
3/22	---	15	5/4	201	6	3/20	---	24	3/19	220	12
3/23	160	10		PIG 5		3/21	269	20	3/20	---	12
3/24	155	10	4/21	280	--	3/22	---	23	3/21	216	17
3/25	150	11	4/22	275	15	3/23	268	19	3/22	---	16
	PIG 2		4/23	280	20	3/24	236	6	3/23	213	11
3/17	175	--	4/24	285	23	3/25	228	8	3/24	208	16
3/18	---	16	4/25	295	10	3/26	209	15	3/25	224	18
3/19	178	16	4/26	296	19	3/27	207	10	3/26	200	10
3/20	167	13	4/27	288	15	3/28	206	11	3/27	198	13
3/21	173	12	4/28	275	12	3/29	199	15	3/28	199	15
3/22	169	15	4/29	271	15	3/30	190	11	3/29	198	12
3/23	---	19	4/30	281	13	3/31	173	10	3/30	181	6
3/24	163	13	5/1	249	15	4/1	161	6	3/31	205	16
3/25	161	13	5/2	242	14	4/2	173	12	4/1	181	12
	PIG 3		5/3	244	19	4/3	163	6	4/2	178	10
3/17	206	--	5/4	233	11		PIG 8		4/3	178	7
3/18	---	13	5/5	229	12	3/17	213	--	4/4	178	8
3/ 19	200	11		PIG6		3/18	---	14	4/5	161	6
3/20	---	6	4/21	234	--	3/19	230	10		PIG 10	
3/21	204	16	4/22	278	15	3/20	---	5	4/21	238	--
3/22	---	16	4/23	241	22	3/21	231	12	4/22	243	13
3/23	201	10	4/24	241	20	3/22	---	22	4/23	240	14
3/24	198	11	4/25	237	19	3/23	242	13	4/24	236	15
3/25	183	12	4/26	246	18	3/24	232	17	4/25	232	13
	PIG 4		4/27	249	21	3/25	224	13	4/26	229	13
4/21	254	--	4/28	228	10	3/26	210	17	4/27	238	--
4/22	250	5	4/29	244	22	3/27	210	14	4/28	242	17
4/23	247	9	4/30	240	12	3/28	212	16	4/29	229	17
4/24	257	25	5/1	234	15	3/29	210	16	4/30	241	24
4/25	263	21	5/2	241	22	3/30	210	17	5/1	231	23
4/26	254	18	5/3	245	14	3/31	200	16	5/2	233	18
4/27	246	10	5/4	237	15	4/1	195	16	5/3	236	14

DATE	BODY-WEIGHT gms	OAT-CAKE gms
5/4	221	13
5/5	220	12
5/6	218	9
5/7	213	13
5/8	206	10
5/9	201	12
5/10	191	11
5/11	182	7
	PIG 11	
4/21	269	--
4/22	265	23
4/23	271	38
4/24	281	23
4/25	285	19
4/26	295	23
4/27	301	19
4/28	304	24
4/29	303	18
4/30	304	20
5/1	295	12
5/2	276	2
5/3	261	5
5/4	249	3
5/5	241	2
5/6	226	2
5/7	213	9
5/8	206	9
5/9	212	5
5/10	209	7
5/11	199	9
	PIG 12	
4/21	258	--
4/22	269	15
4/23	277	20
4/24	271	23
4/25	276	10
4/26	288	19
4/27	297	15
4/28	294	12
4/29	292	15
4/30	309	20
5/1	308	19
5/2	305	20
5/3	317	18
5/4	295	13
5/5	295	19
5/6	299	20
5/7	295	17
5/8	300	23
5/9	298	17
5/10	279	10
5/11	244	2
	PIG 13	
4/21	280	--
4/22	283	13
4/23	275	13
4/24	279	23
4/25	284	28
4/26	293	16
4/27	303	17
4/28	308	16
4/29	306	22
4/30	314	18
5/1	316	21
5/2	327	22
5/3	332	15
5/4	335	17
5/5	341	16
5/6	344	22
5/7	344	19
	PIG 14	
4/21	255	--
4/22	254	13
4/23	260	23
4/24	266	19
4/25	273	18
4/26	279	14
4/27	278	18
4/28	279	17
4/29	278	13
4/30	281	15
5/1	288	20
5/2	297	20
5/3	299	14
5/4	297	15
5/5	301	18
5/6	305	17
5/7	316	17
5/8	313	16
5/9	314	14
5/10	320	15
5/11	325	16
5/12	---	15
5/13	322	15
	PIG15	
4/21	269	--
4/22	265	20
4/23	275	21
4/24	277	24
4/25	274	23
4/26	279	17
4/27	277	18
4/28	278	15
4/29	276	20
4/30	286	18
5/1	290	19
5/2	290	20
5/3	294	14
5/4	292	16
5/5	298	15
5/6	305	17
5/7	310	16
5/8	311	18
5/9	315	14
5/10	316	15
5/11	320	19
5/12	---	15
5/13	322	15
5/14	321	15
	PIG 16	
3/17	225	--
3/18	---	7
3/19	239	7
3/20	---	8
3/21	248	15
3/22	---	16
3/23	259	13
3/24	254	15
3/25	257	15
3/26	256	15
3/27	269	18
3/28	257	7
3/29	271	28
3/30	269	14
3/31	273	18
4/1	263	10
4/2	287	18
4/3	285	15
4/4	284	20
4/5	288	20
4/6	295	22
4/7	299	23
4/8	312	24
4/9	313	12
4/10	322	16
4/11	328	10
4/12	324	10
4/13	341	15
4/14	349	16
	PIG 17	
3/17	207	--
3/18	---	7

DATE	BODY-WEIGHT gms	OAT-CAKE gms
3/19	203	16
3/20	---	10
3/21	199	8
3/22	---	9
3/23	186	3
3/24	180	7
3/25	189	12
3/26	185	18
3/27	191	13
3/28	188	8
3/29	196	14
3/30	200	12
3/31	200	9
4/1	205	12
4/2	206	15
4/3	194	18
4/4	208	14
4/5	206	14
4/6	204	12
4/7	201	12
4/8	214	14
4/9	214	10
4/10	219	10
4/11	228	10
4/12	238	15
4/13	230	14
4/14	232	15